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Emergent evolution but without Mysticism! (Richard Goldschmidt) Systemic Mutations and Saltatory Speciation

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Abstract

The issues of genome architecture rearrangement during speciation are considered. The role of systemic mutations in evolution is analyzed. The author» own phenomenological data on the spatial rearrangement of chromosomal apparatus in the germline tissue (referred to as systemic mutations) during the phylogeny of several dipteran species are systematized. The concept of systemic mutations and their crucial role in saltatory transformation of genomes in the species evolution is further elaborated. The epigenetic mechanisms of speciation, namely, heterochromatin modifications and changes in the spatial organization of chromosomes in germinative cell systems, are considered. The roles of the lamina, topoisomerase II, and polypurine DNA trac in the attachment of chromosomes to the nuclear envelope are discussed. The rearrangement of the spatial organization of chromosomes in the nucleus is postulated as the main event leading to the species-specific fixation of gene mutations, chromosomal mutations, and heterochromatin modifications in speciation. The changes in relationships between chromosomes associated with reorganization of the system of chromosome–nuclear envelope contacts and rearrangement of the chromocenter apparatus in the interphase nucleus are regarded as systemic mutations directly related to speciation. The evolutionary significance of severe inbreeding under extreme environmental conditions (with temperature as a major factor) for formation of adaptive genetic variation and speciation is grounded. The major manifestations of "paradoxical" effect of severe inbreeding are (1) structure function genome reorganization in the generative (reproductive) system and; (2) activation of mobile genetic elements. This can lead to generation of different types of mutations (gene, chromosomal, genomic, and systemic) and heterochromatin modifications. The study of genetic aspects of speciation and adaptation, has revealed several genetic parameters that distinguish between the evolutionarily labile species (with a low level of specialization), which are "generators" of speciation, and the evolutionarily conserved (specialized) species, residing in the terminal segments of phylogenetic lineages. In a horizontal evolutionary development of a taxon (cladogenesis or adaptive radiation), the traits indicating a low genomes specialization are gradually replaced at each step of speciation in the course of progressive special

ization by the alternative traits (evolutionarily conserved), reacheing its maximum manifestation in the terminal species.

Keywords: Emergent evolution, Systemic Mutations

Introduction

The development of evolutionary genetics over the last 50 years has been determined by a shift in focus from routine population genetic studies dealing with gene and chromosome frequencies in populations towards assessment of the evolutionary role of genome structural modifications with regard to their putative saltatory rearrangement [1,2].

Many evolutionary cytogeneticists of the 20th century regarded the genomic and chromosomal rearrangements as an important step in speciation. As for plants, polyploidy was generally considered a universal method of speciation. White (1978) [3], was the brightest protagonist of the concept of chromosomal speciation in animals, and his concept of stasipatric speciation is distinguished among others by its originality. However, the role of chromosomal speciation is not particularly popular among modern evolutionists. The thesis of Dobzhansky (1970) [4], that "homozygotization of two genes is sufficient for a new species to arise" has widely spread among biologists.

This point of view was confirmed by finding the so-called homosequential species, i.e., the species with identical structure of polytene chromosomes, in a several groups of Hawaiian drosophila species [5]. However, the interspecific differences in size and location of heterochromatic blocks in metaphase chromosomes were soon found for some homosequential species [6,7]. Therefore, the problem of chromosomal rearrangement during speciation has remained unsolved until recently, when the phenomenon of systemic mutations was discovered in malarial mosquitoes [8].

Earlier, Goldschmidt (1940) [10], postulated the existence of a special mutation type— systemic mutations- which played the key role in a saltatory origin of various taxonomic groups.

This paper considers how the concepts of («macromutationism has formed») and develops the hypothesis on the essence of systemic mutations and their evolutionary role relying on the own data.

Species-specific remodeling of the interphase chromosome architecture in germinative tissue as a special type of chromosomal mutations associated with speciation

In 1979, the phenomenon of cardinal rearrangement of the architecture of interphase nuclei in the ontogeny and phylogeny of the malarial mosquito was discovered [8], which was later confirmed by the data on drosophila [10,11, 66]. The main difficulty in revealing similar phenomena in other species is the methodological limitations in analyzing the topology of the chromosomal apparatus in germline cells, primarily, in the ovum. Note that it was the analysis of nuclear architecture in germline cells that enabled us to discover the most important regular patterns. On the one hand, we demonstrated the fundamental difference in the chromosome architecture in germline and somatic cells. On the other hand, we found considerable differences in the three- dimensional organization of chromosomes in the germline cells themselves between different (including closely related) species. The species- specificity manifests itself in the following characteristics:

(1) The presence or absence of chromosome contacts with the nuclear envelope and localization of the attachment sites on the chromosomes;

(2) The morphology of the chromosome attachment sites;

(3) Separated attachment sites of homeologous chromosomes of closely related species on the nuclear envelope; and

(4) The presence or absence of the local or diffuse chromocenter.

In other words, both the chromosomes and nuclear envelope carry the local genetically determined areas that account for the contact between the chromosomes and the envelope. Chromosomes may attach to the nuclear envelope with both the centromeric regions and some loci of internal regions. In the sites where polytene chromosomes attach to the nuclear envelope, both the homologs (asynaptic attachment) and the arms

of the same chromosome (at the centromeric regions) can be spatially separated. The contacts between the chromosomes and the nuclear envelope are strictly invariant within each species of malarial mosquitoes and distinctly differ between the all seven studied species. Interspecific hybrids display species-specific characteristics of both species; pronounced spatial separation of the attachment sites of all homeologs in the hybrid cells suggests that the coordinates of the attachment sites on the envelope are species-specific. Therefore, remodeling of the architecture of the interphase nucleus may be regarded as a new type of mutations, which we refer to as systemic mutations [11]. Our interpretation is somewhat similar to the known systemic mutations by Goldschmidt; however, as I will explain below, this analogy is far from being complete and concerns only the phenomenon of "scrambling" of the interphase nucleus.

Systemic mutations result from a spatial rearrangement of the interphase chromosomes in the nucleus because of a change in the chromosome-nuclear envelope interaction. The origin of systemic mutations is associated with a rearrangement of the chromocenter apparatus: the structure of the chromocenter changes (from local to diffuse) until the chromocenter as if disappears, while the centromeres, telomeres, and other chromosomal loci attach to the nuclear envelope or separate from it. The other specific features of systemic mutations are the following:

(1) They are distinctly detectable only in the cells of generative tissue;

(2) They are species-specific and do not display any intra specific polymorphism; and

(3) They display their "heterozygosity" only in interspecific hybrids, the genomes of which reflect the differences in architecture of the "paternal" and "maternal" chromosomes [12].

Presumably, β-heterochromatin, the strands of which enter the nuclear envelope, is the key players in attaching chromosomes to the envelope rather than, α-heterochromatin, which has a compact block structure and does not contact the membrane [13]. In terms of evolution, heterochromatin is the most variable part of the genome and is associated with speciation. Even closely related homosequential species can significantly differ in their heterochromatin content and distribution [6]. Of special interest are the relationships between two Hawaiian species, Drosophila mimica and D. kambysellisi, which are anisohomosequential. D. kambysellisi carries a pair of microchromosomes and D. mimica, a pair of acrocentrics instead of them. Thus, the total amount of heterochromatin in these species is the same, whereas its location is different [14]. A certain differentiation is observed in another anisohomosequential group of Hawaiian fruit flies, comprising D. disjuncta, D. affinidisjuncta, and D. botrucha. These species significantly differ in the amount of heterochromatin in both the autosomes and sex chromosomes. The variations here are not associated with heterochromatin redistribution as in the previous case but rather with the appearance (or disappearance) of additional heterochromatin [15]. A similar situation is also observable in the malaria mosquitoes Anopheles atroparvus and A. labranchiae. The X chromosome of the latter carries a considerably larger heterochromatin block [7,16] reviews numerous data on the interspecific differences in the heterochromatin localization and amount in the Drosophila montium subgroup, the leucosphyrus and maculatus groups of Anopheles, and a number of other Diptera species; all the studied species display distinct differences in heterochromatin although some of them are homosequential. Note in conclusion that our interpretation of the nature of systemic mutations fundamentally differs from the views of Goldschmidt (1940) [9], who believes that systemic mutations are based on the changes in "intrachromosomal pattern (or architecture)" resulting from rearrangements (translocations, inversions, or heterochromatin modifications). On the contrary, we regard the systemic mutations as the mutations that change the relationship between chromosomes or the architecture of the chromosome set as a whole, which is explained by the rearrangement of the chromocenter apparatus and the system of contacts between the chromosomes and nuclear envelope [8]. In the following discussion, I will designate the systemic mutations of R. Goldschmidt as SMG, and the system mutations of V. Stegniy as SMS.

This suggests that homosequential and chromosomally monomorphic species are often stem species and differ from the derived species by that the heterochromatin more frequently resides in the chromocenter, microchromosomes, and sex chromosomes sand less frequently in the euchromatic arms. On the contrary, the species with adaptive inversion polymorphism typically reside at the end of phyletic lineages and their heterochromatin is dispersed over the chromosome arms. Thus, modifications of the heterochromatin components in the genome can be directly associated with chromosome polymorphism and speciation so that any gradual rearrangements on the evolutionarily significant time scale is most likely excluded. Interspecific hybrid incompatibility can be associated with the abnormal mitoses (bridges, cohesions, and ring chromosomes) in early embryo-

genesis resulting from the incompatibility of heterochromatic DNA repeats in closely related species. It is relevant to mention that the sharp differences in heterochromatin were shown to play an important role in maintaining the reproductive isolation between D. melanogaster and it sister species D. simulans [17]. The crosses of D. melanogaster males and D. melanogaster females give normal chromosome segregation in the anaphases of the 10th–13th mitotic divisions in female embryos. However, abnormal mitoses were observed in the hybrid embryos at this stage in the crosses of D. melanogaster males and D. simulans females. When the maternal X chromosomes move to the opposite poles of the spindle, their centromeres are not separated from the paternal X chromosomes, connected via a chromatin bridge. This bridge is heterochromatic in its nature and is represented by a region rich with 359-bp repeats. This results in an incorrect sister chromatid separation in the X chromosome, eventually leading to abnormal mitotic divisions and death of female embryos. The laggings or bridges in the region of 359-bp repeats most likely result from the absence of the corresponding repeats in the D. simulans egg. Along with heterochromatin, one of the most important epigenetic factors in speciation is the chromosome architecture in the interphase nuclei. The chromatin interacts with the nuclear envelope via the contact with laminas and inner membrane proteins, which are part of the nuclear lamina [18]. Lamins can bind to the proteins of chromatin. Thus, the nature of changes in the chromosome attachment to the nuclear envelope as well as a dynamic behavior of the chromocenter in speciation at a molecular level is still vague. The roles of the lamina, topoisomerase II, and polypurine tract [19]. in these processes are obvious; most likely, biophysical approaches will clarify the mechanisms of species-specific spatial chromosome rearrangement. As has been recently shown, the nuclear mechanosensingis implemented via intricate ways ranging from changes in the protein and chromatin conformations and localization of transcription factors to chromosome rearrangement and the membrane dilation up to its rupture, which is often determined by the major structural proteins of the nucleus, lamins [20].

The observed principle of a species-specific reorganization of the interphase chromosome architecture in the germinative tissue is regarded as a phenomenon of a mutational nature, which has no analogs among the known types of chromosomal mutations. This structural reorganization of the interphase nuclei in the generative tissue can be regarded as a new type of mutations, which we refer to as systemic mutations (SMS) [8,11,21]. To better understand how SMS emerge and spread, it is necessary to clarify the notions of heterozygosity and homozygosity in the case of SMS. All individuals of the same species are always homozygous for SMS, whereas the SMS heterozygotes appear in the F1 of the crosses with individuals of another closely related species. Both the female and male F1 progenies are heterozygous for SMS; the important point here is whether they have an adaptation syndrome of a dominant type, i.e., good preadaptation to the environmental conditions of the ecological periphery for the parental species [21]. The individuals of parental species under ecologically marginal conditions display poor adaptive characteristics and yield in fitness to the new forms. The SMS heterozygotes mate each other and homozygous parental type individuals and increase in number. The SMS homozygotes, representing a newly emerging species, appear; if these environmental conditions are optimal for this new species, it rapidly spreads. The newly emerged species is homozygous for all polymorphic gene loci and chromosomal inversions, since all mutations in the genes and chromosomal rearrangements that emerged in parallel with the SMS (or earlier) are immediately fixed in the SMS homozygotes, and the stage of polymorphism, characteristic of heterozygotes, ends with the transition of heterozygotes into a homozygous state. This transient polymorphism lasts for a very short period of several generations (as long as there remain SMS heterozygotes). Thus, SMS as if force the gene and chromosome mutations to be fixed in this new species, which is initially obligatory monomorphic. The new polymorphic variants for both the genes and chromosomes arise later and represent the polymorphic variants of the new species. The genetic monomorphism and species-specificity of molecular mutations discovered by Altukhov and Rychkov [22], as well as the earlier known emergence of species-specific (fixed) chromosomal inversions [23], are explainable by coupling of these mutational events with SMS. The SMS rapidly fix them via passing into a homozygous state, and transfer the genome from one monomorphic state to another. Thus, the transient polymorphism in the genes and chromosomes lasts only for a short period when SMS are heterozygous. The modifications of heterochromatin (changes in the heterochromatin content and location), going on in parallel with SMS, are also rapidly fixed and become species-specific. In essence, SMS and the modifications of heterochromatin in the generative tissue obligatorily coupled with them switch the species genome in terms of the function from one invariant state to another, forming "a new reaction system" according to Goldschmidt (1940). The dominant effect makes it possible to exhibit all the properties of the newly emerging species as early as the first appearing heterozygotes [24]. The transition to a homozygous state is most

rapid, taking just several generations, since heterozygotes being highly viable (as in heterosis) yet have poor reproductive potential.

Hard inbreeding under extreme environmental conditions as the major factor of microevolution and speciation

The emergence of a new species as a result of SMS resolves the problems associated with allopatric (geographic isolation) and sympatric (the absence of geographic isolation) speciation pathways.

In a sympatric speciation, the species distribution range contains ecologically "strained" zones without any geographic isolation, which I define as «ecologically marginal» conditions. The extreme abiotic conditions for a species are observed here, temperature regimes being the most important among them. The role of inbred reproduction is of a paramount importance here and it particularly leads to destabilization of the species genomes, which is putatively associated with formation of either a new species or adaptive genetic polymorphism [25]. In terms of evolution, the emergence of sexual process in eukaryotes was obligatorily associated with the disturbance of random mating (panmixia). In this context, a moderate limitation of panmixia is in fact a common feature of all eukaryotes and is associated with territorial and ecological constraints on individuals and populations within the species distribution range. Considerable constraints on panmixia, such as severe inbreeparthenogenesis, and facultative transition to self-fertilization and self-pollination, are also characteristic of the species with sexual process and most often are associated with drastic changes in the habitat, especially its abiotic factors, with temperature regimes and humidity as the major factors. Inbreeding leads to homozygotization for the alleles of polymorphic genes in the progeny. Hard (brother–sister) inbreeding can be the cause of depression in population. This is a common genetic knowledge.

However, our studies have shown that the inbreeding (especially hard) under extreme temperatures during the ontogeny causes more complex structure–function genome reorganization (primarily affecting the generative system). This reorganization can lead to different evolutionarily significant consequences ranging from emergence of new genetic polymorphism to speciation [21]. The paradoxical effect of hard inbreeding appears as (1) structure function genome destabilization in the reproductive system (in germline cells, the chromosome structure is changed as well as chromatin distribution and content and the chromosome– nuclear envelope contacts appear and disappear) and (2) activation of mobile genetic elements, resulting in "explosive" generation of mutations of different types (gene, chromosomal, genomic, and systemic) and modifications of heterochromatin distribution and content. This has three putative consequences for a species:(1) limitation of the distribution range or death of a species (with ubiquitous and unidirectional environmental changes); (2) further development of a species through formation of adaptive genetic polymorphism owing to newly emerging gene and chromosomal mutations and the corresponding increase in the species distribution range (expansion of the species ecological niche); and (3) emergence of a new daughter species based on the corresponding genomic and systemic mutations (SMS). The spatial organization of chromosomes in germline cells can also considerably vary. Our studies have shown that hard brother–sister inbreeding in combination with low-temperature exposure (cultivation at +16°C) caused considerable modifications of the chromosome apparatus in the nurse cells of the Calliphora erythrocephala fly. In normal laboratory or wildlife populations of this fly, the nurse cell chromosomes are rarely polytene [26] and the nuclear chromatin has a reticular structure. However, the brother–sister inbreeding leads to accumulation of nurse cells with polytene chromosomes in the follicles. These changes, first discovered by Bier [27], increased to the seventh inbred generation (without any selection!) and correlated with the developmental abnormalities in early embryogenesis leading to sterility; note that sterility was not associated with homozygotization of sterility genes since both the effect and the appearance of polytene chromosomes were gradual. The number of such nurse cells and the degree of chromosome polytenization increaseds with each generation; by the 12th–15th generations, they accounted for 30–40% of all nurse cells. If this effect were determined by homozygotization of a certain gene (or group of genes), then all ovarian follicles would similarly change the chromosome structure in nurse cells starting from some generation of inbreeding. However, in fact we observe that destabilization of the normal chromosome pattern gradually increases in the course of inbreeding as well as the number of defective eggs failing to develop into embryos. A similar situation is known for the D. melanogaster strains carrying the otu mutation (pseudonurse cells), which display severe disturbances in the development of ovarian nurse cell chromosomes and, apparently, considerable abnormalities of early embryogenesis leading to the death of homozygotes for this mutation [28]. In our laboratory, the separation of the chromosome 2 arms at the

centromeric region was discovered in the ovarian nurse cells in the case of hard brother–sister inbreeding of the malaria mosquito Anopheles atroparvus [29]. Usually, complete synapsis of the 2L and 2R centromeres of chromosome 2 is observed in wildlife and outbred laboratory populations of this species. Interestingly, the 2L and 2R arms in wildlife populations of the closely related species A. beklemishevi [8], A. labranchiae [30], and A. freeborni [12], are completely separated and attached to the nuclear envelope. The study of cytogenetic effects of hard inbreeding and low temperatures (+16°C) in D. melanogaster germ cells (in the ovarian nurse cell nuclei) showed asynapsis of the homologous chromosomes. Note that the inbred flies maintained at normal and low temperatures differed in the number of asynapses in a statistically significant manner [31].

The studies into spatial organization of the drosophila salivary gland polytene chromosomes during its development under extreme temperature conditions (+15 and +37°C) showed an increase in the frequency of ectopic contacts of non-homologous chromosomes [32], as well as an increase in the chromosome area in the nucleus [33]. In this context, the studies describing the effect of inbreeding on the modifications in the heterochromatin nodules of the pachytene chromosomes in maize meiocytes are of special interest. After two–three generations, the number and size of nodular regions change and chromomeric diffuse structures, ectopic pairing, and paracentric inversions appear instead of usual compact heterochromatin [34,35].

Heterochromatin modifications are described in many Drosophila species [6,7,36]. It is suggested that mobile genetic elements (MGEs) play an important role in such rearrangement of heterochromatin blocks in the genome [37-39]. This hypothesis is supported by the observed differences in the number and location of MGEs in the genomes of D. melanogaster populations of different climatic zones [40]. It is known that the individuals from northern populations, living under extreme conditions (low temperature and inbreeding in small populations), display the changes in the heterochromatin content and location in the chromosomes [41]. Extreme temperature was experimentally demonstrated to have a pronounced effect on the drosophila genome. For instance, the temperature-exposed drosophila strains, influenced by a stepwise changing extreme temperatures (from +29 to +18°C), showed considerable alteration in the MGE localization as compared with the original strain [42,43]. A correlation between inbreeding and genome reorganization is evident in the phenomenon of hybrid dysgenesis. Hybrid dysgenesis is generally described as the result of chromosomcytoplasm interaction. The most common explanation for hybrid dysgenesis, especially the effect of genetic instability of individual loci, is transposition of mobile elements (P and MR factors) present in paternal lines from D. melanogaster wildlife outbred populations into the genotypes of maternal strains after long-term inbreeding. Sved [44], an Australian researcher, suggested another approach to describing the mechanisms of hybrid dysgenesis, not contradicting the first one: Sved explained this phenomenon in terms of spatial chromosome organization. According to this model, hybrid dysgenesis takes place when the genomes of a species are separated for a long time (for example, drosophila laboratory strains and wildlife populations) so that the paternal chromosomes have lost the information necessary for their strict orientation in the zygote.

Extreme abiotic environmental factors are prevalent in the ecological periphery of the species distribution ranges this results in a low population size, leading to inbreeding and enhancing mutational process. On the one hand, a high inbreeding rate in peripheral populations under conditions of environmental stress and the corresponding low migratory activity contribut to an increase in homozygosity of polymorphic genes and chromosomes. In particular, analysis of adaptive chromosomal (inversion) polymorphism in the malaria mosquito A. messeae [45], has shown a clinal patterns of the inversion distribution over the species distribution range; moreover, 2R11 homozygotes segregated and completely prevailed in the populations of the northern part of the distribution range. In this area, extremely low temperatures determined the boundaries of the distribution range (ecologically marginal conditions). On the other hand, new rare inversions unobservable in the other parts of the distribution range appeared in this area. The extreme abiotic environmental factors are prevalent in the ecological periphery of distribution ranges, determining a low size of populations, thereby leading to inbreeding and enhancing mutational process. As is known, the external (ecological) stress caused by the changes in abiotic factors (temperature and chemical agents, both natural and anthropogenic), is of a paramount importance for the existence of populations [46,47]. In addition, certain biotic factors, such as competition, predation, and parasitism, also can induce stress [48]. Although the abiotic and biotic stresses can act independently of each other, these two types of stress often have a synergistic effect because the organisms with the low fitness caused by abiotic stress are more vulnerable to predators and parasites. A number of studies have clarified the role of in-

breeding and its rate in adaptive and evolutionary potential and possible consequences [49-51]. The heat adaptation to climatic factors has been examined in a number of studies on latitudinal and altitudinal gradients [47,52]. Under ecologically marginal conditions, this external (ecological) stress is supplemented by internal (genomic) stress resulting from inbreeding, which increases the synergy and can induce deep genome reorganization [53,54]. Inbreeding itself is a stress factor for the genome and leads to strong destabilization of the organism's functioning by changing the genetic constitution. The phenomenon of inbreeding depression, taking place during the first three to five generations of hard inbreeding, is usually associated with homozygotization of lethal and sublethal mutations and the corresponding drastic decrease in viability and fecundity. As has been shown, inbreeding depression more considerably influences the biological characteristics as compared with the morphological traits [55]. In terms of physiology, inbreeding depression is regarded as a pathological stress, the phenomenon discovered in the mid-1960s by Arshavskii [56], and further developed by Selye [57], in his concept of distress.

Hard inbreeding causes hormonal changes and considerable remodeling of the overall hormonal system, with the corresponding changes in gene activities at the level of regulation [58]. Neurohumoral stress destabilizes the existing morphogenetic system. The hereditary system was shown to respond to stress in an integrated manner relying on the concept of genomic stress introduced by McClintock [59]. with the corresponding mass activation of mobile elements and genome reorganization. Hard inbreeding provokes genomic stress and an explosive MGE activity (transposition bursts). The environmental stress caused by exposure to extreme temperatures and other environmental factors, along with directed selection for domestication of foxes [60,61], and sexual selection in drosophila [62], serves as an active background for such genome reorganizations.

The genomic stress on the background of extreme abiotic environmental factors is enhanced by external environmental stress, which causes structure–function destabilization of the genome, MGE activation, and, as a consequence, the burst of mutability. All types of mutations can occur, including gene, chromosomal, genomic, and systemic mutations (SMS). SMs as the main mechanism of the species genome reorganization result from spatial rearrangement of the interphase chromosomes in the nucleus owing to the changes in chromosome– nuclear envelope interaction, while epigenetic mechanisms (heterochromatin modifications) can provide certain channeling and directionality of mutagenesis [11]. The above arguments substantiating the evolutionary significance of severe inbreeding on the background of extreme environmental conditions of wildlife animal and plant populations can be briefed as follows. The outbred populations of a species in the habitats exposed to environmental stress, which I refer to as ecologically marginal conditions, experience a drastic decrease in the number of individuals. This can take place both at the geographical periphery of the distribution range and within the distribution range. Typically, the limiting factors are abiotic ones (temperature and humidity). The degree of inbreeding increases to the hard brother–sister inbreeding, which eventually transforms in certain organisms into facultative self-fertilization, self-pollination, parthenogenesis, and other ways of mating reduction. The phenomenon of inbreeding depression takes place in the first few inbred generations (typically, three to five); this depression is associated with homozygotization for sublethal mutations. The reduction of genetic polymorphism in inbreeding and almost complete homozygotization of the genome lead to hormonal reorganization in inbred individuals, defined as a pathological stress. Extreme environmental factors, defined as environmental stress, synergistically enhance the effect of pathological stress, and lead to genomic stress in the generative system of organisms. Genomic stress leads to MGE activation as well as the structure function reorganization of the genome, appearing as a sharp increase in mutability and the emergence of gene, chromosomal, and genomic mutations, and reorganization of the chromosome architecture (SMS) in the generative system. This brings about a new genetic polymorphism (gene and chromosomal mutations) that extends the adaptive frames of the species (its ecological niche) and the corresponding extension of the species distribution range or formation of a new species (via genomic and systemic mutations SMS). The adaptive genetic polymorphism can arise only on the basis of gene and chromosomal mutations. As for the genomic and systemic mutations, their emergence leads to the effects associated with speciation [24]. Saltatory speciation involving genomic and systemic mutations (SMS) most likely takes place under critical influence of abiotic and biotic factors, which I define as ecologically marginal conditions [63], This definition generally the definition of environmental stress by Lexer [64].

Thus, the following scenarios of microevolution and speciation are possible in wildlife and under anthropogenic conditions:

Stage I. Formation of the ecologically marginal conditions for a species. The effect of extreme abiotic (temperature, humidity, pressure, etc.) and biotic (predators, parasites, etc.) factors is increased owing to significant ecological and climatic impacts at the boundary of the species distribution range or within this range in ecologically strained zones. A drastic decrease in the population density and size and the corresponding drastic decline or complete cessation of between-population migrations. Transition to inbreeding reproduction;

Stage II. Consequences of the «ecologically marginal» conditions. Inbreeding on the background of extreme environmental factors leads to the reorganization of the hormonal system of organisms and structure function destabilization of the genome of the generative (reproductive) system, including chromatin rearrangement (in both distribution and content) and the appearance - disappearance of chromosome–membrane interactions. MGE activation and explosive generation of different mutations (gene, chromosomal, genomic, and systemic) as well as heterochromatin modifications (its distribution and content); and

Stage III. Emergence of adaptive genetic polymorphism for newly emerging gene and chromosomal mutations or speciation at the expense of emerging genomic or systemic mutations (SMs). SMs rapidly (over one generation) fix gene and chromosomal mutations and transfer the genome from one monomorphic state to another.

Thus, the transient polymorphism of the genes and chromosomes lasts for only one generation [21]. Considering the role of systemic mutations in speciation, it is necessary to emphasize that our interpretation [66], differs from the views of Goldschmidt [65]. In my view, a systemic mutation is not necessarily associated with drastic morphological changes. A species newly formed owing to systemic (as well as genomic) mutations can differ from the initial one only in adaptive physiological characteristics with minimal distinctions in the external morphology, as is observed in sibling species [67-69]. In addition, unlike Goldschmidt, I believe that the systemic mutations (SMS) in germ cells are implemented not individually but rather in a cluster formed by reproduction of oogonia and spermatogonia, which considerably elevates the probability of their fixation. Moreover, natural selection undoubtedly plays a key role at all stages in the establishment of the new species.

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